SUPPLEMENT TO
THE MEDICAL JOURNAL OF AUSTRALIA
Number 4, December 12, 1942.

WAR MEDICINE AND SURGERY

Compiled under the direction of the Committee on the Survey of War Medicine of the National Health and Medical Research Council.

TRAUMATIC SHOCK.

Definition.

It is important to realize the difficulty of defining the word "shock", as used in medicine. Both the word and the condition to which medical people apply it are but vaguely understood, and the confusion from the one partly adds to and partly results from, that due to the other. The original meaning was "a violent collision between particles", but there are now several derived meanings, including a common definition of traumatic shock, namely, "a condition of depressed vitality due to injury". Whatever adequacy this definition has, is largely due to its lack of precision.

The importance of recognizing the difficulty of definition is twofold. In the first place, it emphasizes the fact that shock is not a clear-cut clinical entity. Competent clinicians disagree about its early diagnosis, and those who apply the term freely produce unreasonably good therapeutic results. This is true even if one considers only the more restricted subject of traumatic shock. Grant and Reeve were commissioned to make detailed clinical observations on air-raid casualties, and so surveyed the diagnoses of many surgeons. Their conclusion was that "the grounds for the diagnosis of shock depend rather on the personal views of the individual making it, than on definite and well-established criteria. In our experience, the term is so loosely used in practice as not to give an indication of what we should expect to see at the bedside; it does not tell us how ill the patient is, what symptoms and signs he displays, or what treatment he requires. We prefer not to apply this label."[2] But, unfortunately, a term of one hundred and fifty years' standing is not easily superseded. One can, however, recognize its limitations.

Just as clinicians have been confused by using one term for many stages of a condition, and, indeed, for many different conditions, so have laboratory workers produced experimental shock by various unrelated methods. Moon and his collaborators, for example, believe that there are important differences between the effects of hæmorrhage and of shock, and urge experimenters not to produce shock by means of hæmorrhage.[1] They produced it by placing muscle pulp in the peritoneal cavity, or by injecting histamine subcutaneously, or by inducing anaphylaxis; but the resulting effects are not necessarily comparable with those caused by trauma.

It would be mischievous to claim that the subject of shock is concerned with certain simple issues that have been confused by an unsatisfactory name. But no one can make full use of the available knowledge on the subject if he regards shock as a clear-cut entity. Some workers have reported that their cases of shock received little benefit from serum transfusions; or managed quite well without morphine; or responded quickly to elevation of the limbs; or were benefited by injections of saline solution. None of these departures from standard experience need raise any problem, if it is correlated with the appropriate variation of the shock syndrome. Serum will not revive those who, through lapse of time, have passed into the irreversible stage; morphine is not necessary when pain is absent; elevation of the limbs may readily swing the issue in the patient's favour in early or mild cases; and saline solution will bring great benefit to those who are dehydrated, or those who have had a moderate, recent hæmorrhage.

Functional Pathology of Shock.

The basic conception of the functional pathology of shock is that all serious forms are due to a reduced blood flow to the tissues; the diminished flow is partly due to stagnation, but mainly to a reduction in blood volume. The importance of this idea is threefold: it gives some unity to our knowledge of the various causes of shock, revealing a common factor in such apparently different processes as fright, hæmorrhage and anaphylaxis; it suggests one of the indispensable lines of treatment—the restoration of blood volume; and it provides a theoretical basis on which there has been built a useful conception of a vicious circle operating in shock.

This vicious circle may be represented graphically as follows:

Reduced blood volume → Reduced cardiac output →

↓

Reduced blood pressure → Capillary stasis → Reduced oxygen supply to tissues → Increased capillary permeability → Loss of plasma into tissues → Reduced blood volume.

Perhaps the most that can be advanced critically in favour of this scheme is that none of the steps represented is impossible. A complete demonstration of the interlocking of all the factors has, of course, never been given, and the diagram does not illustrate the sequence of events in one who recovers. The idea of a vicious circle in shock, however, is usually regarded with great favour by clinicians, because it explains what is perhaps the foremost principle in the treatment of shock, namely, the importance of the time factor. Shock is not static; it does not have spontaneous remissions; but, once the compensatory reactions of the body fail, the condition, in the absence

of treatment, moves on inexorably towards death. Each decline paves the way for the next. This is a truth which impresses itself on every clinical observer, and the theory of the "cycle of death" gives him a ready explanation of it.

Reduced Blood Volume.

The idea of a reduced blood volume in shock is so important that consideration must be paid to the various factors, proved, or suggested, to be the cause of it. These factors may act singly or in combination.

1. *Hæmorrhage.*—Frank hæmorrhage is the most obvious cause of reduced blood volume, and concealed hæmorrhage is one of the important diagnostic possibilities to be kept in mind in cases which do not respond to treatment.

In healthy people, an acute hæmorrhage, not exceeding a pint, is tolerated well. Venesections in which one to two pints of blood are rapidly removed, cause the donors to faint in many cases. Nevertheless, it has been found that life can still continue for a few hours even when as much as six or seven pints are lost, and the lives of many such patients can be saved if adequate replacement is made without delay.[5]

After a hæmorrhage, which is going to prove fatal, has been completed, there may be a short period during which the blood pressure remains steady at the reduced value. It then slowly declines to a certain critical level, after which it collapses very quickly. There are thus three phases—compensation, decompensation and collapse.

The methods of compensation for hæmorrhage are: (*a*) increase in pulse rate and in force of heart beat; (*b*) vaso-constriction; (*c*) withdrawal into the blood stream of fluid from the tissues, and of cells from reservoirs such as the spleen; (*d*) increased dissociation of oxy-hæmoglobin; and (*e*) hyperventilation. After a hæmorrhage of one pint, full restoration of blood volume may take twenty-four to seventy-two hours; the return of the red cell count to normal in this case takes some seven weeks.

Three important practical points emerge from a consideration of these compensatory reactions.

The first is that restoration of blood volume is achieved with greater speed than the restoration of the red cell count. This point has been elaborated in the course of numerous experimental investigations, such as those of Peyton Rous and Wilson,[6] who showed that rabbits could be deprived of 75% to 80% of their circulating hæmoglobin without suffering great harm, provided the blood volume was maintained. Similar views are held by modern workers regarding the relative unimportance of hæmoglobin replacement as a life-saving measure after hæmorrhage. For example, it has been stated that, so far as an acute hæmorrhage which has reduced the hæmoglobin to 50% is concerned, "red cells play no vital part in the treatment . . . replacement with plasma gives as satisfactory a result as replacement with whole blood".[5] It is generally accepted that in man the presence of one million red blood cells per cubic millimetre of blood is sufficient to maintain life, provided the total circulating fluid volume is normal. The obvious bearing of these findings on therapy will be discussed later.

The second point is that hæmo-dilution is a characteristic response to hæmorrhage. This is important in that it partly invalidates the use of hæmo-concentration tests to assess the presence and degree of shock.

The third point is that we can no longer accept the view first expressed in 1910 that "a falling blood pressure is the first sign of the onset of shock". Compensatory vaso-constriction may maintain the blood pressure for some time after the shock-producing factors have been in operation, and the fact that an injured person has a normal blood pressure is something to be accepted with only guarded optimism. Moreover, the conception of traumatic shock as something which supervenes hours after the injury, is quite wrong. Shock is a response to injury and begins immediately the trauma is inflicted. It may not become overt for some time. One of the suggestions for the clarification of our ideas on shock is that we should drop the term in favour of a general expression such as "response to injury". We should then visualize the whole process rather than the end result, and we should be more receptive to the new data which are, apparently, still needed to give a complete explanation of the findings.

The question has been raised as to whether hæmorrhage is different from shock in regard to pathology and treatment.[5] There are important differences. Hæmorrhage causes a low blood volume in a subject whose vessel walls have normal permeability; in classical traumatic shock the reduced blood volume is directly due to an increase in the permeability of the capillaries. Therefore, a person with a moderate hæmorrhage is benefited by the administration of normal saline solution, especially if he is dehydrated; shocked patients, on the other hand, lose sodium chloride through their permeable capillaries, and do better when it is not given to them. Again, hæmorrhage is usually followed by hæmo-dilution, whereas traumatic shock frequently causes hæmo-concentration.

It is therefore true that there are definite differences between the reactions to hæmorrhage, on the one hand, and to trauma on the other. Nevertheless, a late effect of a considerable hæmorrhage is to produce classical shock. This effect follows as a secondary result of the reduction in blood volume. As there are numerous factors contributing to the reduction in blood volume in traumatic shock, it would appear that the only reason for separating hæmorrhage from the others is that the effects of hæmorrhage are known with more precision and detail. One is entitled to consider that this reason for the distinction is somewhat inadequate in practice, though it does make for a better understanding of the theoretical issues.

2. *Plasma Loss.*—Burns provide the purest examples of blood volume reduction due entirely to plasma loss. The fluid in the bullæ is a direct exudate and the obvious fluid is just a portion of the whole loss from the blood stream. A similar loss results from mechanical or chemical trauma, and from oxygen lack; and, though less obvious, this loss is just as important. In instances of local trauma, the plasma escapes at first mainly into the injured area. Later the loss may become more general, as the previously uninjured capillary beds become affected by anoxæmia that results from the reduced circulating volume and from the prolonged compensatory vaso-constriction.

3. *Dehydration.*—A person dying of thirst draws on his tissue fluid to provide the water required for insensible perspiration and various metabolic processes. When this source is exhausted, reduction in blood volume occurs, because hydration of the body cells is more important even than maintenance of a normal blood volume.

So far in the consideration of the functional pathology of shock, mention has been made only of those factors which directly reduce the blood volume. There are other factors of some importance.

Psychogenic Factors.

Fear and emotion are well-known causes of circulatory embarrassment. They act through a vagal reflex which causes slowing of the heart rate and decrease in blood pressure. A ready example is that of the person who faints at the sight of blood. The reaction is self-limiting, so that the signs may be alarming but short-lived. These findings suggest, however, that continued anxiety might be expected to aggravate traumatic shock; and it is probably literally true that people have died of fear, through this mechanism.

Neurogenic Factors.

Pain does not always lower the blood pressure, but in certain cases it may do so, by the action, again, of the depressor reflex. A typical example is collapse, following a blow on the testis. Such collapse can be prevented experimentally by blocking the nerves to the part before injury is incurred. A similar type of collapse occurs after the administration of a spinal anæsthetic or after an operation on the brain.

Neurogenic and psychogenic shock have long been classified as primary. It is generally accepted that primary shock usually subsides with, or even without, conservative treatment. Primary shock does not cause reduction of blood volume, but it may be associated with trauma that has this effect. In such cases, the more dangerous element may be revealed only when the patient fails to respond to conservative treatment. If there has been obvious reduction in blood volume, however, one does not wait for this therapeutic test to distinguish primary from secondary shock.

In the light of recent knowledge the subdivision into primary and secondary types of shock seems rather artificial. If shock is a complex response to trauma, primary and secondary shock are different phases of one process. They have been separated, partly in a rather unsuccessful attempt to clarify the ætiology, and partly because of the different type of treatment required. In the so-called primary shock transfusion is not necessary.

Toxæmia.

Surgeons have noted that a patient with gas gangrene infection may sometimes show symptoms of shock, which are relieved when the infected limb is amputated. Toxins of a non-bacterial origin have also been suggested as a cause of shock; the classical one is histamine, but modern reviewers, impressed by the failure to demonstrate this substance in the blood of shocked patients, are prepared to state that "whatever shock is, it is not histamine poisoning".

Other Factors.

Cold is accepted as a contributing factor in shock. Adrenal overactivity, causing excessive vaso-constriction, has also been supposed to be a harmful factor following injury, but there is no conclusive evidence either that the vaso-constriction is excessive or that excessive vaso-constriction would cause shock. Disturbance of adreno-cortical function may help to produce shock. Removal of the cortex causes death from a shock-like condition, and some workers claim that the administration of adreno-cortical hormone helps to restore normal capillary permeability in shocked patients.

Early Diagnosis of Shock.

Early diagnosis is important as being the necessary prelude to early treatment. The following points are considered in making the diagnosis.

1. *The Nature of the Injury.*—Extensive trauma, especially if it results in hæmorrhage, almost invariably produces shock. However favourable the patient's condition seems to be, severe injury is regarded as presumptive evidence of impending shock and thorough anti-shock treatment must be instituted. For example, wounds whose bleeding is of some duration, burns of large surface area, extensively crushed tissues, or wounds following severe dehydration would all come in this class. It is therefore essential to know the nature of the injury, and the clothes should be cut away if necessary to facilitate examination. There has been a tendency of late years to neglect the local lesion and to concentrate on the general condition of injured patients. An accurate knowledge of the local injury, however, is often the best guide to the probable systemic effects.

2. *General Condition of the Patient.*—The amount of sweating, coldness or pain is not a reliable index of the severity of shock, nor is the mental state. Nevertheless, one looks for these and other classical features of shock such as thirst, restlessness, nausea and pallor. The background to the history of injury is also considered, since shock is much more likely following loss of sleep, poor nutrition, marked exertion, dehydration, extremes of temperature or psychologically trying experiences.

3. *Falling Blood Pressure.*—The finding of a low blood pressure is very significant, since a reduction to the so-called critical level of 70 millimetres of mercury is usually found in advanced shock. Taken on the whole, however, this is a late and by no means infallible sign. The blood pressure may be deceptively high in hypertensives, in patients with head injury, and in patients such as children, in whom compensatory vaso-constriction is well marked. Moreover, after spinal anæsthesia the pressure may fall to 50 millimetres of mercury without much immediate danger to the patient.

It should be stressed that blood pressure measurements are used both to diagnose shock and to assess its progress. Their value in diagnosis is now more doubtful in cases with a normal reading, but it is still true that, if the blood pressure has fallen from shock, serial estimations of its further course give a most reliable guide to progress.

4. *Rising Pulse Rate.*—A rising pulse rate is sometimes a useful indication of developing shock, but it is not reliable. It is much less so than the measurement of blood pressure. The volume of the pulse is a more trustworthy sign than the rate.

5. *Hæmo-concentration.*—The hæmatocrit reading or hæmoglobin value almost always rises after shock due to burns, crushing injury or intestinal trauma. In these cases particularly it is a useful diagnostic feature, especially as it occurs before the fall in blood pressure; but it is unreliable in other cases, especially those associated with hæmorrhage.

It should be noted that all of the early signs of shock begin as small deviations from the normal, and steadily progress. Therefore, serial readings of these diagnostic measurements must be taken.

Prevention and Treatment.

In the treatment of shock there are certain simple measures which can be carried out in a first-aid post, or even, in part, at the site of injury. These are: control of hæmorrhage; rest; maintenance of head-down position; supply of warmth; relief of pain; giving of fluids by mouth; and reassurance. These are successful in reviving the simpler cases of neurogenic or psychogenic shock. They are necessary even in severe traumatic shock, but must, in this case, be supplemented by transfusion. This can be given most efficiently at a special resuscitation ward of a hospital. Facilities are also available in a hospital for the administration of oxygen, and for the operative procedures frequently required after successful resuscitation.

Hæmorrhage should be controlled as rapidly as possible, either by pressure or by tying off the bleeding vessel. The tourniquet must be used only as a last resort.

After hæmorrhage has been controlled, the patient must be put at rest. This involves resting the patient as a whole, and immobilizing any injured part of the body as well. A striking example of the benefits of local rest is provided by the results obtained in the last war from the use of the Thomas splint in fractures of the femur.

Rest from mental and emotional strain is also required. Therefore, reassurance is a most important part of therapy. It is supplemented by sedation.

The posture to be assumed during rest is such as to favour venous return. Sometimes a striking improvement is seen in a shocked patient when the limbs are elevated. It has long been customary to raise the foot of the bed of a patient in shock. This sometimes gives definite benefit, and should always be tried, except in cases of chest injury and some cases of head injury.

The idea of warming the shocked patient is based on the known deleterious effects of cold in facilitating the production of shock. The usefulness of the procedure is undoubted, but it has been pointed out that over-heating is a definite danger. Vaso-dilatation of skin vessels withdraws some 500 cubic centimetres of blood to the surface and so removes it from the circulation of vital structures. Moreover, if sweating breaks out, an unnecessary and harmful fluid loss is caused.

Emphasis is therefore placed on the conservation of body heat, rather than the addition of external heat. If an

injured man has been exposed to very low temperatures, it is particularly dangerous to warm him quickly by external heat, since the sudden vaso-dilatation induced may be fatal.

Warming is best secured by the application of blankets and the judicious use of hot water bottles. Electrically heated cages can also be used, but have to be watched carefully. In the case of resuscitation wards in colder climates, it is worthwhile heating the whole room.

Pain is sometimes relieved by efficient splinting and dressings, but usually calls for morphine. Severe pain must be relieved, and recent experience suggests that the average person with such wounds as an extensive burn requires an initial half-grain dose of morphine. It should not be forgotten, however, that morphine has no specific anti-shock value. It is given for the pain, and not for the shock *per se*. Some shocked persons have no pain, and these do not require an analgesic.

Morphine is not always well tolerated; it may induce, or aggravate, nausea or vomiting. In such cases heroin may be effective. One danger in the use of morphine in shock is that a second dose may be given because the first, though adequate in quantity, has been poorly absorbed by the reduced peripheral circulation. Acute morphine poisoning can be caused in this way.

Fluid by mouth, if tolerated, can be given to all casualties, except those with abdominal injury and those awaiting operation. The fluid is best given in frequent, small doses, to avoid vomiting. Water, salted water, coffee and tea are suitable forms.

Whitby has calculated that 10% of patients admitted to hospital as air-raid casualties, need transfusion. Assuming that equipment and a trained staff are available, the chief problems raised are those of the choice of fluid, the amount to be given, and the rate of transfusion.

The chief transfusion fluids available at the present time are saline solution, glucose-saline solution, whole blood, plasma and serum. Gum acacia is sufficiently dangerous to justify the statement that it should no longer be used; similar, but possibly less dangerous, fluids such as pectin or isinglass solutions are also best avoided. Purified albumin solutions have been used successfully, and their high osmotic pressure makes them favourable for the treatment of shock, so that their use may become established in the future.

In treating shock by means of blood transfusion, it does not matter whether the blood is fresh or preserved. In deciding between properly processed plasma or serum, it is immaterial which is used, or whether the product has been preserved in the wet or dry form. These are questions of hospital organization and supply, not of therapy.

Saline solution never gives a permanent effect in established shock, since it escapes from the permeable capillaries. It may even make the condition worse, since it washes proteins out of the blood stream. Saline solution therefore increases the œdema fluid, not the blood volume, in cases of shock. Glucose does this, too, and may have the undesirable action of promoting considerable diuresis as well.

There are two circumstances, however, when an increase in the tissue space fluid is required and when saline solution, therefore, is useful. One is during dehydration, and the other is after a moderate hæmorrhage, when the body has successfully compensated for the reduced blood volume by restoring it at the expense of the intercellular fluid.

Blood is a suitable substitute when blood has been lost. It also serves when the reduction in blood volume is due to plasma loss. The theoretical objection, that blood in such cases causes an unfavourable increase in viscosity, has not withstood critical investigation; so that even large amounts of blood have no harmful effects in such cases, and do good in proportion to their ability to raise the blood volume.

Plasma and serum are ideal for such conditions as burns, and they are effective, as explained earlier, in treating hæmorrhage. Volume for volume they are twice as active osmotically as blood; they make it unnecessary to know the patient's blood group; they are more easily administered; and they can be preserved for long periods. It must not be forgotten, however, that they are expensive to produce, and that some part of the donor's gift is wasted in the process.

The conclusion is, therefore, that, whatever form of shock is present, it is possible to use blood or serum or plasma for successful treatment. In general, however, if supplies were not a limiting factor, one would assess what constituent the patient had lost, and "give blood for blood", and plasma (or serum) for plasma.

The most accurate way of controlling the amount given is to estimate the blood volume. It has been shown experimentally, however, that the blood pressure is a reasonable clinical guide to the sufficiency of a transfusion. A rise of ten to twenty millimetres of mercury can be expected for each pint given. If such a rise does not occur, an unrecognized hæmorrhage should be suspected. It is best to err on the side of giving too much, for a pint or two of fluid will not cause harm to a patient who does not need it. One should aim at the restoration and maintenance of a blood pressure of 100 millimetres at least.

The rate of transfusion can be much faster than in the treatment of non-shocked patients. The early stages may be conducted at a rate of a pint in ten minutes, until the blood pressure is in the region of 90 to 100 millimetres, when the rate should be halved or quartered, and afterwards adjusted according to progress.[6] In some patients "speed" shock may occur and necessitate a reduction in rate.

Improvement usually occurs within a few hours of giving the transfusion, and is sometimes dramatically sudden. It is occasionally delayed, but if there is no material improvement after giving two or three pints to a patient who is not bleeding, then later improvement is unlikely.

Operations required should be performed as soon as possible after the patient is revived. The aim at operation is to do as little as is surgically adequate in as short a time as possible. Blood loss should be kept to a minimum, since shocked patients are extremely sensitive even to small hæmorrhages. Often the transfusion should be continued at a drip rate during an operation, in readiness for the quick replacement of blood, should hæmorrhage occur.

In the case of a mangled limb, operation should be done as soon as transfusion is under way.

Oxygen administration is a useful ancillary method of treatment. Its limitation is that the anoxæmia is not of the anoxic type. Nevertheless, clinical observers have been impressed by its usefulness. It must be administered through an efficient apparatus—a point which has often been overlooked in Australian hospitals.

No discussion of traumatic shock is complete without a reminder that short summaries of the extensive literature are very inadequate. Moon has recently produced a book which gives a fuller treatment of the work done on shock up to the early part of this year.[5]

REFERENCES.

(1) G. A. H. Buttle, A. Kekwick and A. Schweitzer: "Blood Substitutes in Treatment of Acute Hæmorrhage: An Experimental Evaluation", *The Lancet*, Volume II, 1940, page 507.

(2) R. T. Grant and E. B. Reeve: "Clinical Observations on Air-raid Casualties: II", *British Medical Journal*, Volume II, 1941, page 329.

(3) W. d'A. Maycock and L. E. H. Whitby: "Some Aspects of Wound Shock with Experiences in Treatment", *Journal of the Royal Army Medical Corps*, Volume LXXVII, 1941, page 173.

(4) V. H. Moon, D. R. Morgan, M. M. Lieber and D. McGrew: "Similarities and Distinctions Between Shock and the Effects of Hæmorrhage", *The Journal of the American Medical Association*, Volume CXVII, 1941, page 2024.

(5) V. H. Moon: "Shock: Its Dynamics, Occurrence and Management", 1942.

(6) P. Rous and G. W. Wilson: "Fluid Substitutes for Transfusion after Hæmorrhage", *The Journal of the American Medical Association*, Volume LXX, 1918, page 219.